

MEDITATION-BASED TRAINING: A Possible Intervention for Attention Deficit Hyperactivity Disorder

by **SHRUTI BAIJAL** and **RASHMI GUPTA**

AUTHOR AFFILIATIONS: Drs. Baijal and Gupta are from the Centre for Behavioural and Cognitive Sciences, University of Allahabad, Allahabad, India.

ABSTRACT

Recent studies suggest that training-based measures are effective in improving cognitive skills. Meditation-based training has produced lasting changes in brain and cognitive functions. This technique of mental training exhibits neuroplasticity in the attentional networks, exhibiting superior performance, especially in the domain of attention and executive control processing, which is impaired in attention deficit hyperactivity disorder (ADHD). Although intervention techniques for ADHD are well researched, many individuals continue to experience significant functional impairment despite the symptom improvement. This emphasizes a need for a comprehensive approach that requires an effective behavioral intervention. The present paper provides a converging review of meditation-based effects on the brain, dysfunctions of ADHD, and suggestions for enhancement of cognitive abilities in patients with ADHD using meditational training combined with existing measures of intervention. The idea proposed herein should be considered a step for initiation of empirical studies on meditation-based training intervention and outcome effects.



ADDRESS CORRESPONDENCE TO: Dr. Shruti Baijal, Centre for Behavioural and Cognitive Sciences, University of Allahabad, Allahabad, India; Phone: 91-9935256621; Fax: 91-532-2460738; E-mail: shruti_baijal@rediffmail.com

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INTRODUCTION

In adult humans and primates, sensory, motor, and subcortical representations are continually shaped by training and experience.^{1,2} Training-related plastic changes in the brain have shown to occur during various stages of growth and development. Cognitive functions, such as attention, are labile and, therefore, can be altered by training and environmental stimulation. Externally induced processes of development lead to skilled performance in humans, such learning to read or drive a car. This has been accepted by models of cognitive processes that emphasize the role of environmental influences during the development of cognitive functions.³ Thus, certain skilled behavior can train cognitive networks in the brain to produce neuroplasticity. Training-induced neuroplasticity has been found to affect behavior not just at the cortical level,⁴ but also at the level of molecules and genes.⁵

Meditation is a technique of training that has shown a wide spectrum of improvement in cognitive processes and self-regulation abilities.^{6,7} Recently, meditation-based training effects have attracted interest in the scientific community. There has been growing evidence for the beneficial effects of meditation-based training techniques not just in improvement of certain cognitive abilities,⁶⁻⁸ but also in treatment of many conditions, including anxiety disorders, depression, fibromyalgia, chronic pain, substance abuse, binge eating, and skin diseases.⁹⁻¹² This article focuses on contributions from scientific study of meditation and its implications in neuropsychology. First, we will describe the technique of meditation and the underlying cognitive mechanisms that are enhanced due to prolonged meditation training. We will focus on the effects that meditation-based training has on various cognitive processes, such as attention, executive control, and emotion regulation, as well as brain activity and other neurochemical changes. Next, we will discuss the abnormal cognitive and neurobiological functioning in ADHD, and discuss how meditation-based

training possibly may ameliorate the underlying deficits in individuals with ADHD.

EFFECTS OF MEDITATION-BASED TRAINING

Most types of meditation begin with an initial focus of attention and are broadly grouped into two categories: concentrative (focused attention) and mindfulness (distributed attention).^{13,14} *Concentrative* meditation involves focusing attention on a particular object or event, such as one's breathing or a spot on the wall while all other extraneous stimuli are tuned out. *Mindfulness* meditation, on the other hand, involves entering an open state and being receptive to all kinds of stimulation, such as thoughts, emotions, sensations, and images. The two types of meditation may be linked

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to different systems of attention.⁷ Concentrative meditation has been linked to the orienting and conflict monitoring system, proposed by Posner and Petersen¹⁵ as the dorsal attention system,¹⁶ which is described as a voluntary attention system activated by presentation of cues indicating perceptual and response features of stimuli to which participants should direct their attention. In contrast, mindfulness meditation can be linked with the alerting system,¹⁵ or the ventral attention system,¹⁶ which is described as an alerting system that is activated during abrupt changes in sensory stimuli and detection of salient targets, especially when they are unexpected, are outside of the focus of attention, and have low probability of occurrence. Both forms of meditation also share certain common key components, such as body relaxation, positive mood, breathing technique, and mental imagery.

Critical alterations in brain activity occur during the practice of

meditation, which appear as distinct state and trait changes. Trait changes refer to changes in brain activity or behavior—due to long-term practice of meditation—that can be observed even when the person is not meditating.^{6,17} State changes refer to immediate, transient changes during or immediately after a meditation session.⁸ Srinivasan and Baijal¹⁰ showed that the meditators' abilities to automatically detect auditory changes in the environment are enhanced immediately after meditation (increased mismatch negativity or MMN amplitudes) compared to when they simply performed their daily breathing exercises and relaxation controls. The MMN is a difference waveform obtained by subtracting event-related potentials (ERPs) of frequently presented auditory stimuli

from the infrequently presented ones. The amplified MMN for meditators is an indicator of their enhanced sensitivity for novelty detection in the environment. Meditation has been found to be associated with increased regional blood flow or glucose metabolism in prefrontal and cingulate cortex,^{18,19} which has been shown to be involved in cognitive functions of attention and emotions. Some of the transient changes in brain activity due to meditation include bursts of oscillations and increases in global frontocentral coherences, especially in the lower frequency range (i.e., alpha and theta).²⁰ Meditators have been found to self-induce gamma-band oscillations and phase synchronization during meditation, especially in the frontoparietal areas, which signifies an attentive brain.²¹ These changes in brain activity and cognitive processes appear beneficial, thus improving overall cognitive functioning.

The techniques of mental training integrate several key components of

relaxation and mental imagery that show a wide range of positive effects in attention, emotions, social behaviors, and clinical conditions.²² A study on motion-induced blindness and binocular rivalry showed that meditation-based training resulted in certain alterations that allowed

...focused meditation techniques have been shown to reliably facilitate memory for details of an emotionally salient event as demonstrated in a study that compared memory for such events with other memory-facilitating procedures.³²

maintenance of a percept for a very long time,⁶ which is indicative of better sustained attention capabilities. The extensive practice of this technique of stylized attentional deployment exhibits trait improvements in distribution of attentional resources as demonstrated by reduced attentional blink after the meditative training.²³ The reduced brain resource allocation to the first target (T1) in order to leave enough processing resources for the second target (T2) was also reflected by smaller T1-elicited P3b amplitudes. In addition, meditation-based training also caused significant improvement in the attention network showing less distractibility.^{7,17,24} Another important feature of the attentional network is that of response inhibition and conflict monitoring.¹⁵ Prior attentional training through the practice of meditation alters the functioning of dorsal attentional network to improve voluntary response level as well as input level selection processes. The participants who obtained some experience in meditation showed improved performance on the attentional network task.^{7,17} The experienced group was able to detect targets more easily and efficiently even when cues about where and when the targets would appear were unavailable, which is indicative of increased attentional readiness and alertness. Some of these beneficial effects observed with meditation-based training on the attentional tasks are comparable with the positive effects of Mozart music.^{25,26}

Enhanced ability to regulate behavior during an executive control task after training of meditation has also been observed. An event-related potential contingent negative variation (CNV) task requires responding to an imperative stimulus that follows a warning stimulus. Better performance,

as indicated by lower amplitudes in the choice task CNV, was shown by the meditators. This indicated a balanced attentional set and efficient initiation of response preparatory processes. The improved task performance results were supplemented by increased electrophysiological coherence, especially in the frontal areas, which reflects efficient functional coordination of the frontal circuits and thus executive control that enhances self-regulatory processes.²⁷ The frontal striatal pathway has been shown to be affected by practice of meditation. Meditation was associated with increased dopamine release in this pathway, especially the ventral striatum,²⁸ which is linked with monitoring of executive control.²⁹ The practice of meditation gives rise to an acute increase in plasma melatonin levels, which could have health-promoting effects.^{30,31} The effects of the different forms meditation on melatonin levels have yet to be assessed. Therefore, they cannot yet be applied to ADHD and other disorders, particularly for sleep-related malfunctions.

Meditators are also able to better regulate their emotional state, which has been associated with decreased emotional reactivity (increased dorsolateral prefrontal activity). Meditators have been found to show better self-regulation of emotions, as indexed by profile of mood states, enhancing positive mood, and reducing negative feelings.¹⁷ The experimental group after the

mindfulness meditation-based training had a significantly lowered cortisol response to the mental stress after training than did the control group after relaxation training. Meditation activates neural structures involved in attention, such as frontal and parietal cortex, as well as those involved in arousal/autonomic control, such as pregenual anterior cingulate, amygdala, midbrain, and hypothalamus.¹⁹ In addition, focused meditation techniques have been shown to reliably facilitate memory for details of an emotionally salient event, as demonstrated in a study that compared memory for such events with other memory-facilitating procedures.³² Many of the cognitive improvements observed after meditation-based training have implications for the treatment of cognitive impairments in ADHD, some of which will be discussed in the following section.

ABNORMAL COGNITIVE MECHANISMS IN ADHD

ADHD is one of the most common cognitive disorders. For more than 20 years, ADHD has been viewed as comprising three primary symptoms: poor sustained attention, impulsiveness, and hyperactivity.^{33, 34} The behavioral deficits arise relatively early in childhood and remain persistent over development.³⁵ Recent experimental literature reveals that individuals with ADHD have been characterized by specific deficits in monitoring of attentional resources, which in turn negatively affect the cognitive processes, such as response inhibition, error monitoring, attentional disengagement,³⁶⁻³⁸ decision-making processes,³⁹ and emotion regulation.^{40,41}

Attentional processing. There is evidence that the core deficit linked with ADHD involves problems in sustained attention and selective attention functions that are necessary to perform a given task.⁴² These individuals exhibit several deviancies from their developmental level, sufficient to create impairments in major life activities. Most of these impairments have been studied using

objective measures to demonstrate distinct attentional problems. Individuals with ADHD show deficits in subsystems of attention, such as alerting, orienting, and executive network. Individuals with ADHD have been found to demonstrate off-task behavior, becoming less persistent on the given task, increased vulnerability to distraction, inability to cope with the change in rules of a task, and being less capable of regulating attention when dealing with more than one task.⁴³⁻⁴⁵

Some of the sustained attention problems among ADHD individuals may also be linked with deficits in alerting mechanisms,⁴⁶ which are critical for normal cognitive functioning. Earlier work using spatial-orienting tasks suggested that ADHD individuals show difficulty in maintaining the alert state (sustained attention) in the absence of a warning signal.⁴⁷ More recent studies using the Attentional Network Task (ANT) have replicated problems with alerting in ADHD, again mostly due to the inability of the individual to maintain the alert state when no warning signal was used.^{48,49} Other studies using tasks similar to ANT have also shown some evidence of abnormalities in alerting and/or executive control in ADHD in terms of slowed response times to abrupt visual cues, especially when faced with conflicting spatial cues.⁵⁰ The attentional impairments in ADHD also result in suppression of some event-related potential components, such as N2 for discriminating frequent and infrequent targets in an auditory and visual oddball task; Nd for discriminating attended and non-attended stimuli, and P3 for discriminating target and non-targets in choice reaction time tasks (for review see ref. 42).⁴² It should be noted that impairment in attention processes also results in abnormal functioning in many higher-order cognitive operations that involve inhibition of a pre-potent response, interference control, and emotion regulation, some of which will be discussed later in this article.

Executive control. A number of studies provide consistent empirical

support for the assumption that individuals with ADHD have a deficit in executive-control processes, one of which is response inhibition.^{51,52} ADHD individuals normally show slower responses during a stop signal task,⁵³ which requires responses to “go” trials and inhibition of responses during “no go” trials. Earlier, it was suggested that the ADHD individuals are deficient at monitoring responses during “no go” trials. However, meta-analytic results have shown potential milder problems during “go” responses as well, indicating that problems in response organization and arousal may also play a role in the disorder.^{52,54} In addition, a functional magnetic resonance imaging (fMRI) study that compared neural activation while contrasting “go” and “no-go” trials showed greater activation of ventrolateral prefrontal cortical areas that subserve response inhibitory processes in particular, as well as anterior cingulate and frontopolar regions that are implicated in other executive functions.⁵⁵ These results are consistent with the problems of response inhibition that are observed in ADHD individuals while performing a flanker task. More specifically, the impairments in executive-control processes manifest as poor post-error slowing and decrement in accuracy when faced with response conflict during a flanker task. Such deficits in monitoring ongoing behavior during the task were confirmed by the reduced event-related potential amplitude difference

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between correct and incorrect trials compared to normal controls.^{56,57} A recent fMRI study indicates a functional anatomical asymmetry of response inhibition between ADHD and normal children.⁵⁸ Response inhibition in normal children was

related to premotor activation in the right hemisphere while in the ADHD children it was associated with robust activation of the right posterior superior temporal cortex. This region has been associated with control of actions⁵⁸ and may reflect alternative, perhaps compensatory, processes in ADHD.

Cognitive performance of individuals with ADHD is also characterized by large moment-to-moment fluctuations in cognitive control reflected by a highly inconsistent and inaccurate response style. It has been suggested that abnormal error processing underlies this failure to implement adequate control that provides top-down adjustment of elementary mental operations.³⁶ ADHD individuals slow down after inhibition failures only in a few trials, and the magnitude of slowness was less compared to normal subjects. The normal subjects slow down after the error trials most of the time and also to a greater extent. This suggests that normal subjects are able to better adjust their behavior after errors. Another study also elaborated on error monitoring as one of the executive-control measures impaired in ADHD using the event-related potential (ERP) methodology. The error-related negativity (ERN) was the same for ADHD and the control; the former showed diminished error positivity (Pe). Based on these findings, they concluded that individuals with ADHD are normal in

early-error-monitoring processes related to error detection as indexed by ERN, but show abnormal response-strategy adjustments and are deviant in later error-monitoring processes related to Pe, which may also be associated with the

subjective/emotional, conscious evaluation of the error.⁵⁹ This is also an indication of the abnormal emotional regulation in these individuals. Yet in another study, brain electrical activity was recorded during the stop-signal task. Individuals with ADHD showed abnormal scalp distribution in P3a and reduced ERN in dorsal anterior cingulate cortex, suggesting a global deficit in cognitive control operations in these individuals.⁶⁰ ERP amplitude differences between correct and incorrect responses were also found to be diminished in individuals with ADHD. ADHD individuals also show larger deficit in control processes necessary for disengagement from one task and preparation for a subsequent task. It has been suggested that behavioral deficits observed in individuals with ADHD are the result of deficient resource-allocation policies. Individuals with ADHD have problems adjusting their speed in order to cope with higher-task demands, which suggests an under-arousal and poor state regulation.⁶¹

ADHD individuals are also characterized by the abnormalities in reward responsivity that interferes with decision making. A recent positron emission tomography study suggested that neural circuits engaged during decision making differ in subjects with ADHD when compared

the finding that ADHD individuals are characterized by a specific motivational style called *delay aversion*.⁶³ Delay aversion is the tendency to avoid a delay in results by taking small, immediate rewards over large, delayed rewards. This is an indication of an abnormal emotional/motivational mechanism in individuals with ADHD.

Neurobiology. Several investigations have found ADHD to be associated with abnormal levels of neurotransmitters and hormones. It is well known that alerting depends heavily upon norepinephrine,⁶⁴ while the conflict monitoring, motor movements, and ability to focus attention on a task involves dopamine.⁶⁵ These are the two transmitters most often implicated in ADHD. In ADHD, low levels of dopamine disrupt abilities of maintenance of attention on a task, resulting in switching from one activity to another due to lack of focus. A number of animal models have tried to explain the putative neural substrates of altered dopamine function. One of the leading models is that of rats selected for familiar hypertension (spontaneously hypertensive rats or SHR) featuring hyperactivity, impulsivity, and poor sustained attention. The anomaly with one or the other type of control in one or more

mesostriatal projections. This may in turn reflect reduced release, mediated by D2 receptors.⁶⁷ It provides an explanation for the poor inhibitory control that depends on frontostriatal circuitry in rats and humans.^{68,69}

Some other neurobiological problems in ADHD may account for a good number of ADHD patients who experience chronic sleep-onset insomnia.⁷⁰ The inability to sleep at the desired time may be due to imbalance in melatonin levels due to its delayed release,⁷¹ and such problems may disrupt cognitive processes while performing everyday tasks. Melatonin-based treatments have been shown to improve circadian rhythms of sleep-wake cycle and ameliorate sleep-related abnormalities.⁷¹ Medication-based treatments may bring melatonin levels to normalcy, but may not enhance any cognitive performances.⁷¹ There are, however, other methods of training that can produce similar or better effects.³¹

IMPLICATIONS OF MEDITATION-BASED TRAINING IN TREATMENT OF ADHD

A number of interventions exist for treatment of ADHD, which include pharmacotherapy,⁷² cognitive behavioral therapy,⁷³ and educational/physical/speech therapy.⁷⁴ Although none of these treatments eliminate all symptoms of ADHD in all patients, some techniques may have an edge over others. Certain medications have been found to facilitate improvement in the daily functioning of these individuals. Medication treatments may help patients with ADHD to mobilize the effort required to improve their cognitive functions, and medication is sometimes more effective than behavioral treatment measures.⁷⁶ For some patients, however, medication does not improve cognitive skills or their performance in school, employment, or social relationships.⁷⁵

A study by the MTA Cooperative Group⁷⁷ showed that the existing behavioral techniques for the treatment of ADHD are inadequate

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to normal subjects. Ventral and dorsolateral prefrontal cortex and the insula were activated during performance of the decision-making task in both the ADHD and normal groups; however, activation in the ADHD group was less extended and did not involve other regions, such as anterior cingulate and hippocampus that subserve emotion/memory processes. This difference may explain observed deficits in motivated behaviors in individuals with ADHD.⁶² This observation is also consistent with

dopamine-projection systems in the SHR may be nonlinear and occur during early development. The binding sites for dopamine release may differentially affect activity in the mesolimbic, mesocortical, and mesostriatal projections. There are reports of reduced dopamine (dopamine agonist or DA) release for the SHR mesostriatal projection.⁶⁶ The DA dysfunctions fail to modulate excitatory glutamatergic and inhibitor GABAergic neurotransmission in the mesolimbic, mesocortical, and

and may not even point to the symptoms of ADHD. The behavioral treatment adopted by the MTA group did not effect improvement in ADHD symptoms but affected the non-ADHD characteristics of the patients.

However, if one considers ADHD to be a malfunction of cognitive processes, then fostering purely medication-based treatments may not be sufficient in its treatment. A recent

these approaches do not undervalue the importance of the support provided by a parent, mentor, clinician, or therapist. Also, due to the presence of different styles of meditation, it is yet to be explored as to which form of meditation may be more effective in ameliorating cognitive impairments. Moreover, it should be noted that the spectrum of cognitive improvements observed in the normal population may

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study of the effects of meditation on adolescents and adults with ADHD showed improvement in the domain of attentional processes and symptoms of the disorder.⁷⁷ Although the study was limited by a small sample size and lack of control group, it suggested that using meditation-based training in addition to other existing interventions in patients with ADHD may be beneficial.

The research on meditation-based training and its effects suggests that meditation may enhance certain attentional capabilities.^{8-10,23} Meditation has successfully been used to reduce symptoms of stress, anxiety, chronic pain, and depression.¹¹⁻¹⁴ These improvements may be due to the functional as well as structural plasticity of the brain systems²⁷ that result in neurophysiologic state changes, which eventually evolve into trait effects secondary to long hours of practice, stylized attentional deployment, reframing of cognitive context, and emotional regulation involved in meditation training.⁷⁸

The variety of cognitive deficits that interact during ADHD and the variety of cognitive improvements that are observed with the proposed meditation-based training procedures are consistent with the idea of integrated neural circuitry underlying various cognitive functions.⁷⁹ This focuses on the common, unified brain operations that do not require a need for the existence of any specialized, functional cortical areas. Of course,

not be fully applicable to individuals with ADHD. Thus, there are limitations in the nature and magnitude of improvements in cognitive functions that can be expected with this training procedure. With this perspective, we propose that the future research on interventions of ADHD should include employing meditation-based training measures in controlled, longitudinal studies. It is also important that psychiatrists receive training in meditation so that they can augment their practices with this important technique.⁸⁰

REFERENCES

1. Buonomano DV, Merzenich MM. Cortical plasticity: from synapses to maps. *Ann Rev Neurosci* 1998;21:149-86.
2. Wang X, Merzenich MM, Sameshima K, Jenkins WM. Remodelling of hand representation in adult cortex determined by timing of tactile stimulation. *Nature* 1995; 378:71-5.
3. McClelland JL, Rogers TT. The parallel distributed processing approach to semantic cognition. *Nat Rev Neurosci* 2003;4:310-22.
4. Maguire EA, Gadian DG, Johnsrude IS, et al. Navigation-related structural change in the hippocampi of taxi drivers.[see comment]. *Proc Natl Acad Sci U S A* 2000;97(8):4398-403.
5. Meaney MJ. Maternal care, gene expression, and the transmission of individual. *Annu Rev Neurosci* 2001;24:1161-92.

6. Cahn BR, Polich J. Meditation states and traits: EEG, ERP, and neuroimaging studies. *Psychol Bull* 2006;132:180-211.
7. Srinivasan N, Bajjal S. Meditation: Brain activity and cognitive changes. In: Sangeetha Menon (ed). *Consciousness, Experience and Ways of Knowing: Perspectives from Science, Philosophy and the Arts*. Bangalore: NIAS, 2006:155-83.
8. Carter OL, Prest IDE, Callistemon C, et al. Meditation alters perceptual rivalry in Tibetan Buddhist monks. *Curr Biol* 2005;15:412-13.
9. Jha AP, Krompinger J, Baime MJ. Mindfulness of training modifies subsystems of attention. *Cogn Affect Behav Neurosci* 2007;7:109-19.
10. Srinivasan N, Bajjal S. Concentrative meditation enhances pre-attentive processing: Mismatch negativity study. *Neuroreport* 2007;18(16):1709-12.
11. Astin JA. Stress reduction through mindfulness meditation: Effects on psychological symptomatology, sense of control, and spiritual experiences. *Psychother Psychosom* 1997;66:97-106.
12. Kabat-Zinn J, Massion AO, Kristeller J, et al. Effectiveness of a meditation- based stress reduction program in the treatment of anxiety disorders. *Am J Psychiatry* 1992;149:936-43.
13. Kristeller JL, Hallett CB. An exploratory study of a meditation-based intervention for binge eating disorder. *J Health Psychol* 1999;4:357-63.
14. Marlatt GA, Kristeller JL. Mindfulness and meditation. In: Miller WR (ed). *Integrating spirituality into treatment: Resources for practitioners*. Washington, DC: American Psychological Association, 1999:67-84.
15. Posner MI, Petersen SE. The attention system of the human brain. *Annu Rev Neurosci* 1990;13:25-42.
16. Corbetta M, Shulman GL. Control of goal-directed and stimulus-driven attention in the brain. *Nat Rev Neurosci* 2002;3:201-15.
17. Tang YY, Ma Y, Wang J, et al. Short-term meditation training improves

- attention and self-regulation. *Proc Natl Acad Sci U S A* 2007;104(43):17152–6.
18. Newberg A, Alavi A, Baime M, et al. The measurement of regional cerebral blood flow during the complex cognitive task of meditation: A preliminary SPECT study. *Psychiatry Res* 2001;106:113–22.
19. Lazar SW, Bush G, Gollub RL, et al. Functional brain response to relaxation mapping and meditation. *Neuroreport* 2000;11:1581–5.
20. Aftanas LI, Golosheikine SA. Non-linear dynamic complexity of the human EEG during meditation. *Neurosci Lett* 2002;330:143–6.
21. Singer W. Neuronal synchrony: A versatile code for the definitions of relations? *Neuron* 1999;24:49–65.
22. Walsh R, Shapiro SL. The meeting of meditative disciplines and western psychology: A mutually enriching dialogue. *Am Psychol* 2006;61(3):227–39.
23. Slagter HA, Lutz A, Greischar LL, et al. Mental training affects distribution of limited brain resources. *PLoS Biol* 2007;5(6):e138.
24. Brefczynski-Lewis JA, Lutz A, Schaefer HS, et al. Neural correlates of attentional expertise in long-term meditation practitioners. *Proc Natl Acad Sci U S A* 2007;104(27):11483–8.
25. Ho C, Mason O, Spence C. An investigation into the temporal dimension of the Mozart effect: Evidence from the attentional blink task. *Acta Psychologica* 2007;125:117–28.
26. Thompson WF, Schellenberg EG, Husain G. Arousal, mood, and the Mozart effect. *Psychol Sci* 2001;248–51.
27. Travis F, Tecce JJ, Guttman J. Cortical plasticity, contingent negative variation, and transcendent experiences during practice of the Transcendental Meditation technique. *Biol Psychol* 2000;55:41–55.
28. Kjaer TW, Bertelsen C, Piccini P, et al. Increased dopamine tone during meditation-induced change of consciousness. *Brain Res Cogn Brain Res* 2002;13:255–9.
29. Koepp MJ, Gunn RN, Lawrence AD, et al. Evidence for striatal dopamine release during a video game. *Nature* 1998;21266–8.
30. Massion AO, Teas J, Hebert JR, et al. Meditation, melatonin and breast/prostate cancer: Hypothesis and preliminary data. *Med Hypotheses* 1995;44:39–46.
31. Tooley GA, Armstrong SM, Norman TR, Sali A. Acute increases in night-time plasma melatonin levels following a period of meditation. *Biol Psychol* 2000; 53(1):69–78.
32. Wagstaff, GF, Cole J, Wheatcroft J, et al. A componential approach to hypnotic memory facilitation: Focused meditation, context reinstatement and eye movements. *Contemp Hypnosis* 2007;24(3):97–108.
33. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Third Edition*. Washington, DC: American Psychiatric Press, Inc., 1987.
34. Barkley RA. *Hyperactive Children: A Handbook for Diagnosis and Treatment*. New York: Guilford, 1981.
36. Logan GD. On the ability to inhibit simple thought and action II. Stop signal studies of repetition priming. *J Exp Psychol* 1985;11:675–91.
37. Rabbit PM. Three kinds of error-signalling responses in a serial choice task. *Q J Exp Psychol A* 1968;20(2):179–88.
38. Schachar R., Chen S, Logan GD, et al. Evidence for an error monitoring deficit in attention deficit hyperactivity disorder. *J Abnorm Child Psychol* 2004;32:285–93.
39. Garon, N. Decision making in children with ADHD only, ADHD-anxious/depressed, and control children using a child version of the Iowa Gambling Task. *J Atten Disord* 2006; 9:607–19.
40. Maedgen JW. Social functioning and emotional regulation in the attention deficit hyperactivity disorder subtypes. *J Clin Child Psychol* 2000;29:30–42.
41. Walcott CM. The relation between disinhibition and emotion regulation in boys with attention deficit hyperactivity disorder. *J Clin Child Adolesc Psychol* 2004;33:772–782.
42. Greenham SL. Attention-deficit hyperactivity disorder and event-related potentials: evidence for deficits in allocating attentional resources to relevant stimuli. *Child Neuropsychol* 1998;4:67–80.
43. Borger N, van der Meere J. Visual behaviour of ADHD children during an attention test: An almost forgotten variable. *J Child Psychol Psychiatry* 2000;41:525–32.
44. Hoza B, Pelham WE, Waschbusch DA, et al. Academic task performance of normally achieving ADHD and control boys: Performance, self-evaluations, and attributions. *J Consult Clin Psychol* 2001;69:271–83.
45. Lorch EP, Milich M, Sanchez RP, et al. Comprehension of televised stories in boys with attention deficit/hyperactivity disorder and nonreferred boys. *J Abnorm Psychol* 2000;109:321–30.
46. Posner M, Raichle M. *Images of Mind*. New York: Scientific American Library, 1994.
47. Swanson JM, Posner MI, Potkin S, et al. Activating tasks for the study of visual-spatial attention in ADHD children: A cognitive neuroanatomical approach. *J Child Neurol* 1991;6:S119–27.
48. Blane MN, Marrocco R. Cholinergic and noradrenergic inputs to the posterior parietal cortex modulate the components of exogenous attention. In: Posner MI (ed). *Cognitive Neuroscience of Attention*. New York: Guilford, 2004:313–25.
49. Booth J, Carlson CL, Tucker D. Cognitive inattention in the ADHD subtypes. Paper presented in the 10th meeting of the International Society for Research in Child and Adolescent Psychopathology, Vancouver, Canada. 2001.
50. Oberlin BG, Alford JL, Marrocco RT. Normal attention orienting but abnormal stimulus alerting and conflict effect in combined subtype of ADHD. *Behav Brain Res* 2005;165:1–11.
51. Barkley RA. Behavioral inhibition, sustained attention, and executive

- functions: Constructing a unifying theory of ADHD. *Psychol Bull* 1997;121:65–94.
52. Sergeant JA, Oosterlaan J, van der Meere J. Information processing and energetic factors in attention-deficit/hyperactivity disorder. In: Quay HC, Hogan AE (eds). *Handbook of Disruptive Behavior Disorders*. New York: Kluwer Academic/Plenum, 1999:75–104.
53. Schachar R, Logan G. Are hyperactive children deficient in attentional capacity? *J Abnorm Child Psychol* 1990;18:493–513.
54. Oosterlaan J, Logan GD, Sergeant, JA. Response inhibition in ADHD, CD, comorbid ADHD+CD, anxious and control children: A meta-analysis of studies with the stop task. *J Child Psychol Psychiatry* 1998;39:411–26.
55. Schulz KP, Fan J, Tang CY, et al. Response inhibition in adolescents diagnosed with attention deficit hyperactivity disorder during childhood. *Am J Psychiatry* 2004;161:1650–7.
56. van Meel CS, Heslenfeld DJ, Oosterlaan J, Sergeant JA. Adaptive control deficits in attention-deficit hyperactivity disorder (ADHD): The role of error processing. *Psychiatry Res* 2007;151(3):211–20.
57. Wang B, Sui MQ, Wang YF, Fan J. A preliminary study on the attentional networks of attention deficit hyperactivity disorder. *Beijing Da Xue Xue Bao* 2004;18;36(4):370–3.
58. Vaidya CJ, Bunge SA, Dudukovic NM, et al. Altered neural substrates of cognitive control in childhood ADHD: Evidence from functional magnetic resonance imaging. *Am J Psychiatry* 2005;162:1605–13.
59. Leube DT, Knoblich G, Erb M, et al. The neural correlates of perceiving one's own movements. *Neuroimage* 2003;20:2084–90.
60. Wiersma JR, van der Meere JJ, Roeyers H. ERP correlates of impaired error monitoring in children with ADHD. *J Neural Transm* 2005;112(10):1417–30.
61. Liotti M, Pliszka SR, Perez R, et al. Abnormal brain activity related to performance monitoring and error detection in children with ADHD. *Cortex* 2005;4:377–88.
62. Wu KK, Anderson V, Castiello U. Attention deficit hyperactivity Disorder and working memory: A task switching paradigm. *J Clin Exp Neuropsychol* 2006;28(8):1288–306.
63. Ernst M, Kimes AS, London ED, et al. Neural substrates of decision making in adults with attention deficit hyperactivity disorder. *Am J Psychiatry* 2003;160:1061–70.
64. Antrop I, Stock P, Verté S, et al. ADHD and delay aversion: The influence of non-temporal stimulation on choice for delayed rewards. *J Child Psychol Psychiatry* 2006;47:1152–8.
65. Marrocco RT, Davidson MC. Neurochemistry of attention. In: Parasuraman R (ed). *The Attention Brain*. Cambridge, MA: MIT Press, 1998:35–50.
66. Madureira DQ, de Carvalho LA, Cheniaux E. A neurocomputational model for the thalamocortical loop: Towards a better understanding of attention deficit hyperactivity disorder. *Arquivos de NeuroPsiquiatría* 2007;65:1043–9.
67. Oades RD, Sadile AG, Sagvolden T, et al. The control of responsiveness in ADHD by catecholamines: Evidence for dopaminergic, noradrenergic, and interactive roles. *Dev Sci* 2005;8(2):122–31.
68. de Jong W, Linthorst AC, Versteeg HG. The nigrostriatal dopamine system and the development of hypertension in the spontaneously hypertensive rat. *Arch Mal Coeur Vaiss* 1995;88:1193–6.
69. Aron AR, Fletcher PC, Bullmore ET, et al. Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nat Neurosci* 2003;6:115–6.
70. Eagle DM, Robbins TW. Lesions of the medial prefrontal cortex or nucleus accumbens core do not impair inhibitory control in rats performing a stop-signal reaction time task. *Behav brain res* 2003;146(1):131–44.
71. Corkum P, Rimer P, Schachar R. Parental knowledge of attention-deficit hyperactivity disorder and opinions of treatment options: Impact on enrollment and adherence to a 12-month treatment trial. *Can J Psychiatry* 1999;44:1043–8.
72. van der Heijden KB, Smits MG, Gunning WB. Sleep-related disorders in ADHD: A review. *Clin Pediatr* 2005;44(3):201–10.
73. Mészáros A, Czobor P, Bálint S, et al. Pharmacotherapy of adult attention deficit/hyperactivity disorder (ADHD): A systematic review. *Psychiatr Hung* 2007;22(4):259–70.
74. Ramsay JR. Current status of cognitive-behavioral therapy as a psychosocial treatment for adult attention-deficit/hyperactivity disorder. *Curr Psychiatry Rep* 2007;9(5):427–33.
75. Patel K, Curtis LT. A comprehensive approach to treating autism and attention-deficit hyperactivity disorder: a pre-pilot study. *J Altern Complement Med* 2007;13(10):1091–8.
76. Brown TE. *Attention Deficit Disorder: The Unfocused Mind in Children and Adults*. New Haven, CT: Yale University Press, 2006.
77. MTA Cooperative group. A 14-month, randomized, clinical trial of treatment strategies for attention deficit hyperactivity disorder. *Arch Gen Psychiatry* 1999;56:1073–86.
78. Zylowska L, Ackerman DL, Yang MH, et al. Mindfulness meditation training in adults and adolescents with ADHD: A feasibility study. *J Atten Disord* 2007 Nov 19 [Epub ahead of print]
79. Davidson RJ. Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *Am Psychol* 2000;55:1196–214.
80. Fuster JM. *Cortex and Mind: Unifying Cognition*. New York, NY: Oxford University Press, 2003.
81. McGee M. Meditation and psychiatry. *Psychiatry* (Edgemont) 2007;5(1):28–41. ●